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MILK AND SCARLATINA.

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Milk-borne epidemics of scarlet fever have been reported with more or less frequency ever since the English, during the latter half of the last century, called the attention of the medical profession to this method of spreading the scarlet-fever contagion. Such epidemics have been traced in some instances to the exposure of milk to infection from scarlet-fever patients, in others to milk from cows suffering from a disease supposedly analogous to scarlet fever in human beings. From the first the English have been the ones to lay the greatest stress on the agency of milk in carrying infection, as can be seen from the fact that of 330 epidemics of infectious disease, traced to contaminated milk, no less than 243 have been recorded by English authors (Kober).[1] The usual reason given for this preponderance of milk-borne epidemics in England is that Anglo-Saxons are the only people who drink raw milk to any extent; other European peoples habitually heat or boil their milk (Freeman,[2] Kober, Hall,[3] Jürgensen[4]). It is true that America has reported only 52 epidemics as against the 243 of England, but this is said to be due to laxness of sanitary supervision of the milk supply in this country, and lack of careful search for causes in the presence of epidemics (Freeman). As further proof of the agency of milk in disseminating scarlet fever, many authors point to the fact that countries in which milk is not used as an article of diet, or very sparingly used by children, are free from scarlet fever. Thus the disease is practically unknown among the Japanese and very rare among the Hindoos.

In reading the literature on this subject one is impressed with the difference in the attitude of Continental and of British writers. While the former admit that apparently milk is a good culture medium for the virus of scarlet fever, and that there is evidence of infection having been conveyed from one human being to another through this agency, yet their attitude toward specific instances of such milk-borne epidemics is critical, if not skeptical. They insist that before charging the milk with the responsibility of carrying the contagion, other more usual methods must be ruled out, and there must be clear proof of the relation of the milk to the spread of the disease. The responsibility of milk in the spread of scarlet fever cannot be regarded as proved beyond reasonable doubt, unless the outbreak in question presents the following features:

The epidemic must be either distinctly localized or in scattered but sharply defined foci. It must be sudden in its origin and subsidence. The persons affected in the early days of the epidemic must be drinkers of milk from a certain supply. Attendance at the same school, play-grounds, Sunday-school, etc., must be excluded. It must be shown that the milk was exposed to the contagion of scarlet fever, and the epidemic should subside after the supply of infected milk is cut off. Direct sources of infection should be ruled out, as when disease is carried by the boy who delivers the milk, or when the buyers of the milk come in direct contact with the patient in the shop.

Very few reports have proof of this character, most of them failing to exclude sources of contagion other than the milk supply. Harrington[5] gives an interesting illustration of the erroneous conclusions which may be arrived at by insufficient probing of the evidence. In an outlying part of Boston, in 1897, a sudden epidemic of scarlet fever broke out among the children of well-to-do families. Nearly all the families took milk from the same dealer. No fault could be found with the dairy farm; the cows were healthy, and there was no scarlet fever among the dairy workers. The fact, however, that all of the cases appeared in families using the same milk pointed strongly to this as the source of the contagion. Had the inquiry stopped there this epidemic would have been added to the long list of milk-borne epidemics, but a more careful search revealed the fact that the first case of all appeared in a family which had milk from another source; that this child had played with and infected other children, who had spread the disease still farther. The children belonged to the same social set and were playmates. "Thus, what might have served as a most useful example of a milk-borne epidemic fell to the ground."

As an instance of a carefully studied and critically framed report may be quoted Tingvall's[6] account of an epidemic in Westeras, Sweden. During 1901 there were only 27 cases of scarlet fever throughout the whole city. In January, 1902, within four days, 27 cases developed in 22 families living in the same part of the city. No case of scarlet fever had occurred in this region in recent times. The stricken families had nothing in common except the source of their milk supply. Eighteen families bought milk from a dairy farm almost daily; three more bought it occasionally, and almost all drank it uncooked. The one remaining family had bought milk from a farm where there had been scarlet fever shortly before. Two cases of scarlet fever were discovered in the house where the employés of the dairy farm lived. After January only 11 cases of scarlet fever developed in the whole city during the rest of the year, and none in that part of the city.

Another instance, reported as early as 1870 by Oscar Home Bell,[7] leaves nothing to be desired in the way of evidence. He describes an outbreak of scarlet fever which appeared first at St. Andrew's University; then, after the university closed for the holidays, and the milk formerly supplied to it was distributed in the town, the disease appeared in the town. The wife of the dairy farmer had a sore throat and two of her children had typical scarlet fever. She nursed them, and, at the same time, milked the cows and handled the milk. The households in which the cases occurred were, with one exception, houses supplied from this farm, and all the households taking the milk had scarlet fever except one, in which there were no children. Two boys, who had already had scarlet fever, called at the farm regularly for milk and carried it home, as a result of which three other children in the family, who had not visited at the farm, took scarlet fever.

Another careful report is that of the Rostock epidemic given by Jürgensen,[4] who is able to state that those drinking the milk boiled escaped infection. Certain of those cited by Freeman[2] are also satisfactory, as are some in Kober's[1] list, many in Hart's[8] first tabulation, some in his second,[9] Wende's[10] account of the recent Buffalo epidemic, and Eckholm's[11] account of an epidemic in Wasa.

On the other hand, the English journals are full of reports which ignore ordinary rules of evidence, and which incline one to suspect that the theory of the agency of milk in disseminating scarlet fever has taken so strong a hold upon that country that, in the absence of direct proof to the contrary, an English health officer is apt to assume that any localized epidemic of this disease is traceable to contaminated milk. The pages of the *Lancet*, of the *British Medical Journal*, and of *Public Health* abound in such reports as the following: "An outbreak of scarlet fever in Clapton. Dr. J. King Warry was able to associate the outbreak with the distribution of a particular milk supply, but he could find no evidence as to the manner in which the milk became infected. There was no reason for thinking that the milk was infected from a human source."[12]

"Mr. Henry Armstrong has a careful report of an outbreak of scarlatina, apparently associated with the use of milk from a certain dairy. Of the 19 cases 17 took milk from the same dairy. There was some suspicion of scarlet fever in the family of a dairy worker some few months before the outbreak, but there were no other cases among the 219 other families taking the same milk."[13]

"Dr. Wallace regards 8 out of 26 cases in October as clearly connected with this contaminated milk, and 4 out of 21 cases in November as unquestionably due to the same cause." This cause was "unrecognized cases of scarlet fever in the person of the milk dealer and his child," who had suffered from "cold and sore throat." The cases were not treated medically.[14]

"In one street 21 families (more than half those supplied with the milk) yielded 29 cases; some streets taking the milk suffered no attacks. The active principle of contagion was therefore apparently not evenly distributed."[15] "Exciting cause of epidemic: Illness, suggestive of scarlatina, in the families of the two millmen supplying the implicated milk, upward of three months prior to the outbreak."[16] "Epidemic sudden and cessation well marked; of the 205 households invaded, 74, or 34 per cent., were supplied with milk from one farm. Exciting cause of outbreak not ascertained."[17] "Scarlatina, which had hitherto chiefly affected the poorer quarters, suddenly blazed out in better-class houses exclusively, and three-quarters of the homes invaded dealt with one dairy. Cows in a febrile condition, due doubtless to the unsanitary character of their housing accommodations. A sample of mixed milk of several cows showed bacteria (character not given)."[18]

In strong contrast to this attitude are the unenthusiastic utterances of German and of some American writers:

"Observations have recently been made which indicate that it (the scarlet-fever contagion) may be conveyed in milk" (Osler[19]). "The possibility is not excluded that scarlatina can be transmitted by means of the milk" (Jürgensen[4]). "In England it has been thought that the contagion may be carried by milk" (Strümpell[20]). "Without examining into the trustworthiness of the published reports of epidemics of scarlet fever attributed to the milk supply, I would simply state that it is extremely difficult to produce indubitable proof of such occurrences so long as we are in ignorance of the specific cause of the disease. However, the mere possibility that so malignant a disease may be communicated in this way deserves our full attention" (Eckholm[11]). "The question should not be regarded as definitely disposed of, but is one that merits the most searching investigation by pathologists and bacteriologists" (Kober[21]).

This difference between English observers and those of other countries cannot be explained simply on the ground that milk-borne epidemics of scarlet fever are more common in England than in other countries. Undoubtedly, the fact that the English drink their milk unboiled explains why there should be a greater number of milk-borne epidemics of infectious diseases there than in Germany, France, Austria, or Italy, but does not explain the enormous difference between their record and that of the Scandinavians, who also use unboiled milk. Kober[1] gives the list for Scandinavia as 11, as against the 243 of England, and Eckholm[11] speaks of the milk-borne epidemic of scarlet fever at Wasa, in 1903, as only the third thus far reported in Sweden.

In all the epidemics thus far considered the contagion is supposed to have passed from a case of human scarlet fever to the milk, and to have been carried by the milk to a healthy individual, who thereupon contracted the disease. Admitting this to be not only a possible but a fairly frequent mode of infection in scarlet fever, what shall we say as to that second class of milk-borne epidemics—those, namely, in which the disease is supposed to originate in the cow, and the contagious virus to be present in the milk at the time of milking?

The English are practically the only adherents of the theory that scarlet fever is a disease of cattle and can be conveyed from them to human beings. Their literature is full of references to outbreaks of scarlet fever and diphtheria originating in the milk of diseased cows. The first epidemic attributed to "what we now confidently recognize as scarlatina in the cow" (Hart[9]) broke out in 1885, and an investigation, ordered by the London Local Government, was carried out by W. H. Power, Klein, Cameron, and Wynter-Blythe.[22] As a result, the disease was found to be limited to the users of the milk from a dairy farm at Hendon. W. H. Power was convinced that the milk drunk by these scarlet-fever patients came from certain cows which were suffering from a vesicular and ulcerative eruption of the udder. This eruption was directly communicable from the cows to the hands of the milkers, and from them again to other cows. Klein isolated from the vesicles of both cows and milkers a streptococcus which, when injected into calves, produced in them visceral lesions resembling those of scarlet fever in man, namely, congestion of the lungs, parenchymatous degeneration and congestion of the kidneys, and hemorrhages in the spleen. He also isolated the streptococcus from the milk of the diseased cows, and from the blood of 4 out of 11 cases of scarlet fever, which streptococci he used to produce a local vesicular eruption in calves. Therefore, he concluded that this organism is the cause of scarlet fever in man, and of this particular ulcerative eruption in cows. Cameron gave a detailed description of the disease among the Hendon cows, calling it a specific, contagious, constitutional disease, capable of being communicated to man. A milker received the virus into a recent scratch on the forefinger. General weakness, malaise, loss of appetite resulted, and, after about four or five days, a vesicle or small blister appeared on the finger; later, several on the back of the hand. The whole hand became swollen and inflamed, the inflammation extending in broad lines as far as the elbow. The general disturbance lasted a fortnight. Cameron considered the disease quite distinct from cowpox. Calves inoculated with scrapings from the ulcers developed vesicles at the site of inoculation.

The report of Power, Klein, Cameron, and Wynter-Blythe aroused much comment, and from this moment we find the attention of British physicians turned to the now famous "Hendon cow disease" as a frequent cause of epidemics of scarlet fever in man. The literature of epidemics originating in the use of milk from diseased cows increases rapidly, and it soon becomes unnecessary to show that the disease in question has the characteristics of the "Hendon cow disease." It is sufficient to state that the cows were suffering from an "obscure febrile condition;"[23] that they were "off their feed, giving ropy milk, and had scales on the teats"[24] or "abraded teats,"[25] or a rash over the body consisting of "small, red pimples."[26] Excrementitious matter or vaginal discharge may also cause contamination of the milk by the virus of scarlet fever. Thus, Power, quoted by Hart,[9] finds the origin of certain cases of scarlet fever in the use of milk from a cow who had "lost portions of her coat," and whose "buttocks and posterior udder were fouled by excrementitious matter and, perhaps, by vaginal discharge as well." Sewage-polluted water drunk by the cows, or used to wash milk-cans, is also supposed to be responsible for scarlet fever in the drinkers of the milk. Scarlyn Wilson[27] considers the discovery of "bacteria" in milk as confirmatory proof that the milk of cows kept in filthy surroundings caused scarlet fever in children, and the *British Medical Journal* makes respectful editorial comment on his report. The enthusiasm for this theory of the direct causative relation between a diseased condition in cows and scarlet fever in drinkers of their milk becomes so great that, in 1897, we find W. H. Power and Ernest Hart[9] not only accepting all the reports up to date, but adding many still more wonderful. Thus, Hart discusses the question whether a certain epidemic of scarlet fever was caused by the rinsing of milk-cans in sewage-contaminated water or by the use of milk from a cow newly calved, and inclines to the latter belief. The fact that the milk of newly calved cows can cause scarlet fever is stated by Hart to be "an important discovery of modern times," due largely to the researches of Power. Apparently the same kind of milk can cause different diseases. Thus, "ropy milk" caused an outbreak of diphtheria in Hendon in 1883, of scarlatina in Charlton in 1892, and of typhoid fever at Warminster in 1895. It is distinctly stated by Hart that the disease among the cows at Glasgow, which was supposed by Russell to be responsible for an outbreak of diphtheria in that place, was the same as the "Camberwell cow malady," which was considered by Sir George Buchanan and by Klein to be the cause of an epidemic of scarlet fever. The milk of newly calved cows caused scarlatina in London in 1883, at Macclesfield in 1889, and at Sutton Coldfield in 1891; while at York Town and Camberley in 1886 the same sort of milk caused diphtheria, and in Dundee in 1889 it was the "vera causa of infective quality of milk causing typhoid fever." Ernest Hart cites all these reports and concludes: "Thus, we are face to face with a fact, and it is the more important when we remember that the condition attaching to newly calved cows is not one which has been deemed of any importance by veterinary inspectors. Indeed, we may be pardoned for saying that the subject of milk-borne disease is furnishing a history of long struggle by medical as opposed to veterinary opinion. But this struggle has been harder in the matter of eruptive diseases of cows than on any other point, and the victory has certainly been with the medical experts."

Nor are these extreme views confined to one or two writers. In 1896 we find Thorne-Thorne[28] stating that the milk from diseased cows can communicate scarlet fever to man. Editorials in the *Lancet*, as late as 1892, add their testimony to the same effect, and so do the editorials in the *British Medical Journal*, as late as 1894.

Not all of the British authors, however, and practically no one in countries outside of England, hold the views outlined above. (See Allbutt's *System*, vol. iii. p. 163.) At the time of the government investigation of the famous Marylebone epidemic there were voices raised in dissent to the conclusions reached by Klein and Power, but the dissenters, being chiefly veterinarians, did not command much respect from the medical profession. The Agricultural Department of the Privy Council was roused by the statements of Power and his colleagues to make a searching inquiry into the disease among the Hendon cows, which was said to have caused scarlet fever in man. They entrusted the inquiry to Edgar Crookshank,[29] whose report is careful and convincing, and shows a singularly modern attitude toward the bacteriological aspect of the question. Crookshank had an opportunity to investigate an epidemic among cows which occurred in another county, but which was apparently the same as that just previously seen at Hendon, corresponding in every respect to the description of the latter and being communicable to calves and to milkers by direct inoculation. From the lesions on the cows' udders and on the hands of the milkers he isolated Klein's streptococcus. But among the 1500 to 2000 drinkers of the milk from this herd there was not one case of scarlatina, and neither here nor at Hendon did the disease which the milkers contracted resemble scarlet fever. He studied the lesions on the hands of the milkers and concluded that they were those of "typical Jennerian cowpox," and that the irritation and abrasion in the process of milking was responsible for the difference in appearance of the ulcers on the cows from normal pocks. Lymph taken from the human vesicles caused typical vaccinia in calves.

Crookshank concluded that Klein's streptococcus is identical with that found in diphtheria, erysipelas, puerperal fever, scarlatina, etc., and that when inoculated into animals it sets up simply a septic inflammation, not a specific infectious disease. The lesions produced experimentally by Klein pointed only to sepsis, not to scarlatina. When this streptococcus is found in the blood of scarlet-fever patients it should be regarded as a complication, not as causally related to the scarlet fever. Other veterinarians, as Brown and MacFadyen, concurred in this report. Brown[30] stated that a disease, identical in the opinion of veterinarians with that at Hendon, prevailed in parts of eleven counties in which no scarlet fever was found. The outbreak at Hendon was, therefore, not an isolated one, and was elsewhere not associated with scarlet fever in man.

In Germany and in America the general opinion held is that the "Hendon cow disease" was cowpox, or something closely allied to it, and that the scarlet-fever epidemic was only coincident, or that it was not true scarlet fever, but a septic fever due to the presence of pus in the milk. Careful examination of the reports, unfortunately often very meagre, of cases of scarlet fever caused by the drinking of milk from diseased cows brings to light certain features which suggest a disease other than typical scarlatina. Baginsky,[31] for instance, comments on the mild character of the disease in the Hendon epidemic, only 1 fatal case among 60, and the short incubation period of two days. The statement as to a one to two-day incubation period is also made by C. N. Cooper.[32] Clothier,[33] in his account of the outbreak at Stroud Green, says: "As has been observed in previous epidemics of scarlatina disseminated by milk, the type of the disease is mild, and no deaths are reported."

Harrington[34] abstracts the report of an epidemic in Lincoln, England, in 1903, involving 40 persons, the majority of whom were seized on the same day and none of whom was seriously ill. There was a remarkable susceptibility to infection in this epidemic, no less than 14 persons in one household suffering from the disease, a fact which certainly would in itself incline one to doubt the diagnosis of scarlet fever. The milk to which this attack was attributed came from cows with vesicles on the udders. In an epidemic recorded by G. Buchanan[35] the persons attacked with what was held to be scarlet fever, 19 in number, were all adults. Cream of which all had partaken was held responsible for the outbreak, but there was no evidence that any case of scarlet fever had come in contact with it.

Now, if we assume that the milk in these instances was infected, not with the specific virus of scarlatina, but with pyogenic cocci from the inflamed udders of the cows, the matter becomes clear at once. That mammitis in the cow is accompanied by the appearance of streptococci and staphylococci in the milk has been proved by many investigators. Guillebeau[36] examined the milk of 76 cows with inflammation of the udder and found that all contained pyogenic cocci virulent for animals. Nocard and Mollerau[37] demonstrated streptococci in the milk of cows with mammitis, as did also Adametz,[38] Hueppe[39] and others, to whose researches must be added those of Klein, cited above.[29] Bergey[40] found that streptococci and staphylococci persist in the milk for some time after all external signs of mammitis have subsided. At the height of the inflammation these germs are present in enormous numbers. The descriptions of the condition of the cows in the Hendon epidemic, and in others resembling it, show that pus, scales, and blood must have found their way into the milk through the friction of the milkers' hands. Dr. Russell[41] found cocci in the milk of a cow suffering from the Hendon disease, and also in the blood of the calf she was suckling. In other cases it is apparent that the cows were suffering from a catarrhal mammitis at the time, or shortly before the epidemic among drinkers of the milk. The theory of Power as to the infectious nature of the milk of newly calved cows may have some foundation in the greater tendency to mammitis just after calving, or possibly the cows may have been suffering from a mild form of puerperal sepsis.

There are, in fact, two or three reports of cases of throat inflammations attended with constitutional symptoms, which apparently came from the drinking of milk containing pus. Kober[21] describes two epidemics of follicular tonsillitis in a boys' school, with severe gastric disturbances; one involving 50 and the other 84 cases. The cows showed the lesions of variola vaccinia. In 1869 Fagan[42] reported a case of pseudomembranous stomatitis following the drinking of milk contaminated with pus and blood from an abscess in the udder of the cow. Gray Edwards[43] describes with much care and accuracy some cases of follicular tonsillitis due to infected milk. Cultures from the throats of the 12 cases and from the milk yielded the same organisms, namely, staphylococcus aureus and albus and streptococcus. Three children who drank only boiled milk escaped the infection. The "ropy," or "stringy," or "slimy" condition of the milk mentioned by some authors, and especially emphasized by Power and Klein as the cause of scarlet fever and diphtheria, is a condition caused by a wide variety of organisms, including certain strains of staphylococcus pyogenes (Passet, Lehman, and Neumann), and various other cocci and bacilli.*

* Bac. lactis sapon. (Weigmann and Zirn, Centralbl. f. Bakt., Bd. xv. p. 463); Micrococcus Freudenreichii and Bacterium Hessii (Guillebeau, Centralbl. f. Bakt., 1892, Bd. xi. p. 439); Bacillus lactis viscosus (Adametz, Centralbl. f. Bakt., 1891, Bd. ix. p. 698); Leischmann's bacillus (Centralbl. f. Bakt., Bd. xvi. p. 122). Reference to still others is made in Adametz's original article (Berliner Landw. Jahrb., 1891), cited above. Guillebeau's micrococcus Freudenreichii is considered by Lehmann and Neumann as probably a streptococcus. Neither his nor Adametz's organisms were pathogenic.

It is evident that milk-borne epidemics of scarlet fever do occur, but there seems no reasonable basis for the belief that they can originate in a diseased condition of the cow. In this connection we cannot do better than quote the words of Kober.[21] "When we consider that toxins may and do produce a scarlatinous exanthema, we feel warranted in saying that in all the epidemics of scarlet fever and diphtheria which were traced to milk from cows suffering from inflammatory lesions of the udder or from puerperal fever we have typical instances of a streptococcus and staphylococcus infection. . . . There is no proof that there is a disease in the cow which is communicable as scarlet fever to man."

In view of our ignorance as to the infectious agent in scarlet fever, it is interesting to know that milk serves as a good culture medium for it. Most authentic reports of this kind show that the milk was directly exposed to the contagion, as when the milking or handling of the milk or washing of the cows was carried on by persons suffering from a mild form of scarlet fever, or in the stage of desquamation following it. In many instances, however, the contact is less direct; a healthy person apparently carries the infection from a case of scarlet fever to the milk. Wives and daughters of dairymen nurse cases of scarlet fever and handle the milk at the same time. Some of these reports must undoubtedly be taken with caution, for the possibility is not excluded that the selling of the milk was carried on in the room where the sick person lay, or the delivery made by a person ill with scarlet fever, but in others the evidence of indirect infection is very clear. Of Hart's 99 epidemics of milk-borne scarlet fever, the disease was said to exist either at the dairy or on the milk farm in 68. In 17 of these, persons actually suffering from or recovering from scarlet fever handled the milk, but in others the connection was less direct. Two were supposedly caused by bottles or cans left in infected houses; 1 by the wiping of cans with an infected cloth; while in 3 the milk, though not handled by the patient, was stored in or close to the sick-room. In 6 instances dairy employés lodged in or had visited infected houses. Whittaker[4] gives an instance of infection of the milk and of the milkman's daughter through rags stored in the house and used to wipe the cans. The girl came down with the disease in another town on the day following her departure from home.

Many authorities on scarlet fever treat with a certain suspicion the instances given in the literature of infection through a third person, himself free from the disease. As the scarlet-fever virus is not highly infectious, and is apparently confined to the air immediately around the patient, many are inclined to believe that the carrying of the infection by third persons is very rare. But if, as seems very probable, milk is a good culture medium for this virus, it is easy to see why it should figure prominently in such indirect infection. Kerschensteiner[45] says that if infection is to take place through a healthy third person the "scarlet fever dust" must be blown to that person, must cling to his skin, hair, or clothes, and must be blown off from him to the third person; and as scarlet fever is not very infectious, a large amount of the infectious stuff is needed. Now, if we imagine the virus not carried in a dry condition and inhaled as dust, but planted in a liquid culture medium and then, after a lapse of time sufficient for its growth, brought in contact with the throat in the process of swallowing, it is easy to believe that infection through the medium of a healthy third person can take place with comparative frequency, and also that utensils exposed to the air of the sick-room, even at a distance from the patient, may become infected sufficiently to carry the disease.

CONCLUSIONS. The literature of milk-borne epidemics of scarlet fever contains many which are (*a*) based on insufficient evidence as to the agency of milk in the dissemination of the disease; (*b*) based on the erroneous belief that a disease in cows is capable of causing scarlet fever in man.

The disease in cows supposedly responsible for scarlet fever in man is ordinary cowpox, and the disease in man supposed to be caused by it is either septic fever from infection from pyogenic organisms or coincident scarlet fever.

There is no good evidence that milk from diseased cows can cause scarlet fever in man.

After rejecting many reports of milk-borne outbreaks of scarlatina there remains a fair number which are above criticism, and which apparently prove that milk is a good culture medium for the scarlet fever virus, and is the most frequent agent of indirect infection in this disease.

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THE CAUSE OF PULSATIONS IN EMPYEMA.

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GUILLIAM DE BAILLON in the sixteenth century reported the first authentic case of pulsating empyema.

Le Roy (1776) speaks of the possibility of pulsating empyema in his Aphorisms, 489: "When the pus sac lies on the heart and large vessels in such a manner as to transmit their movements there develop false impressions of an aneurysm."

Berard (1836), Stokes (1838, first used the term pulsating empyema), MacDonnell (1844, first used the term empyema necessitatis pulsans), Walshe (1851), Wintrich (1854), Flint (1856), Aran Heyfelder, Owen Ress (1858), Schoostek (1865), Gourbon (1870), and Muller Plagge, Traube (1872), Frentzel (1875), Topham and Dunin (1878) each report one case.

Traube thinks the pulsations are due to a pericardial effusion associated with the empyema.

Comby (1881), reviewing the literature, reported a case and explained the pulsation by saying: "When the lung is completely collapsed, pushed to one side and adherent to the pericardium, it no longer resists the heart, the pulsations of which are then transmitted to the fluid in the pleural cavity, thence to the thoracic wall."